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ACUTE HEMATOGENOUS STREPTOCOCCIC PERITONITIS.

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Definition. Acute hematogenous streptococcic peritonitis is the designation used by the writer in cases produced by a streptococcus infecting the peritoneum by way of the blood stream without an apparent intermediary lesion of an abdominal viscus being demonstrable.

Primary Peritonitis. Authorities are divided as to whether a true primary peritonitis exists. Medical literature is meager in reports. Lewi,[1] Meade,[2] Collie[3] and Semple[4] described several

[1] Tr. Med. Soc. New York, 1873, p. 143.
[2] British Med. Jour., 1876, ii, 393.
[3] Med. Exam., 1877, ii, 843.
[4] Ibid., 1878, iii, 553.

cases and attributed the peritonitis to exposure. Grawitz,[5] in 1886, reported 13 cases of "idiopathie" peritonitis. In 1893 Tavel and Lanz[6] divided the peritonitides into a primary group and other numerous secondary groups classified according to the viscus harboring the causal focus. Flexner,[7] in 1898, simplified the classification by subdividing all secondary cases into an exogenous and endogenous type, depending on whether the infection reached the peritoneum directly from without (*e. g.*, operation, stab wound, lesion of the abdominal wall) or from a lesion of an abdominal organ. The primary cases he attributed to infection carried to the peritoneum through blood or lymph channels.

In 1901 Nothnagel[8] denied the possibility of such an entity as primary peritonitis. In a case in which streptococci were present in the throat and peritoneal exudate, he claimed that the organism had been swallowed with the sputum and wandered out through the intact stomach wall thus reaching the peritoneum. Sheldon[9] believed these cases had their origin in microscopic lesions of the appendix or other abdominal organs. Armstrong[10] insisted on the Fallopian tubes being the seat of the primary infection, because his cases of primary peritonitis all occurred in females. Wilder[11] believed they were secondary to throat lesions. Bondi[12] and Jensen[13] maintained that microscopic intestinal lesions could initiate many of these primary cases of peritonitis.

Jensen fed some animals with virulent cultures of pneumococci in capsules. One of the animals died. At the postmortem examination a purulent peritonitis was found. A follicular enteritis with slight necrosis of Peyer's patches was present, but there was no ulcer or perforation. Pneumococci were found in the intestinal canal, intestinal wall, blood and peritoneum. Flexner,[14] in 1895, mentions the finding of diplococci in the lumen of the intestine, mucosa, submucosa and within the peritoneum in certain cases of infection of the intestine. These organisms were also present in lymphatic spaces, which he believed were the avenue of transit into the peritoneal cavity. He,[15] however, stated that while it would be possible for organisms to wander out through the normal intestinal wall, no definite proof of such an occurrence had yet been offered. Flexner believed that in all cases wherein the intestine was responsible a gross pathological lesion was present. Abacoumova[16] reports a case

[5] Charité Ann., 1884, xi, 779.
[6] Mitt. a. klin. u. med. Institutou d. Schweiz, 1893, i, 1.
[7] Philadelphia Med. Jour., 1898, ii, 1019.
[8] Wien. med. Presse, 1901, xlii, 1321.
[9] Med. Rec., New York, 1902, lxii, 394.
[10] Montreal Med. Jour., 1903, xxxii, 728.
[11] Tr. Chicago Path. Soc., 1916, x, 46.
[12] British Med. Jour., 1905, ii, 232.
[13] Arch. f. klin. Chir., 1903, lxix, 1134; Ibid., lxx, 91.
[14] Bull. Johns Hopkins Hosp., 1895, vi, 94.
[15] Flexner: Loc. cit.
[16] Thèse. de Lausanne, 1905.

in which streptococci were present in the intestinal wall. Lennander's[17] case was one of follicular enteritis.

The theories of gastric or intestinal causation appear disproved by the reports of cases of primary peritonitis in which the causative organism has at an early stage been recovered from the blood. Künzel's[18] case, in which most thorough gross, microscopic and bacteriological studies were made at the postmortem examination, furnishes us with absolute evidence as to the direct infection of the peritoneum by way of the blood stream from the nasopharynx without an intermediary abdominal lesion. Appendix, spleen, intestine, uterus, tubes and ovaries were all normal. On culture these organs proved sterile, or, as in the case of the intestine, yielded no organisms of the streptococcic group. The streptococcus was recovered only in the nasopharynx and peritoneal exudate.

Type of Primary Peritonitis. The type of primary peritonitis mentioned by Flexner, Manahan[19] and Fishbein[20] occurred exclusively as a terminating infection in chronic disease—*e. g.*, valvular defects, chronic parenchymatous nephritis, arteriosclerosis, cirrhosis of the liver and visceral carcinomata. The writer has studied 8 cases of primary peritonitis, all due to the streptococcus, that were entirely of a different character. Seven occurred in females and only one in the male. Their ages ranged between ten months and twenty-one years. They were in the best of health when stricken with this disease. In no instance could the peritonitis be ascribed to a metastatic process in an abdominal organ the result of a septicemia or pyemia. Metastatic foci in abdominal organs are rare in cases of general streptococcic infection.[21] This is offered as an explanation for the absence of any other abdominal lesion in this series. This is quite the opposite with staphylococcic infection, the presence of pyemic visceral processes being the rule. Ehringer,[22] in 1906, collected 21 cases of a similar type to that mentioned by the writer. Holt,[23] Martin,[24] Monks,[25] Bonnet,[26] Oppenheimer[27] and Dowd[28] have each recorded cases since Ehringer's report.

We believe that an acute inflammation of the nasopharynx, particularly of the tonsils, served as the original focus from which virulent organisms passed into the blood stream, reached the peritoneum and through their activity set up a violent peritonitis. We know that the peritoneum is, as a rule, unusually resistant to infection. What lowers the resistance of the peritoneum in these cases?

[17] Upsala Läkaref. Forh., 1900, vi, 248.
[18] München. med. Wchnschr., 1904, li, 1920.
[19] Boston Med. and Surg. Jour., 1905, cli, 346.
[20] Am. Jour. Med. Sc., 1912, cxliv, 502.
[21] Monographic Medicine, 1910, ii, 185.
[22] Thèse de Paris, 1906.
[23] Arch. Pediat., 1900, xxiii, 268.
[24] Ann. Surg., 1900, xliv, 917.
[25] Ibid., 1908, xlvii, 994.
[26] Lyon Méd., 1900, cvii, 821.
[27] Deutsch. Ztschr. f. Chir., 1900, lxxxiii, 456.
[28] Ann. Surg., 1908, xlviii, 821.

Shall we in accordance with the view that has been expressed by Rosenow claim that this particular type of streptococcus has a selective affinity for the peritoneum? Animal experimentation by Chapelle[29] in 1907 failed to establish any such selective affinity.

RELATIONSHIP TO EPIDEMIC STREPTOCOCCIC SORE-THROAT. Some dozen or more epidemics of septic throats are on record.[30] In those of recent years bacteriological studies have invariably proved a streptococcus as the offending organism. Capps[31] described it as a hemolytic capsulated streptococcus. Davis and Rosenow[32] found it to be a peculiar streptococcus. Later, Davis[33] acknowledged its close relationship to the Streptococcus hemolyticus and thought that the two were probably identical. This fact is of extreme importance, for although our cases did not occur during an epidemic of streptococcic sore-throat, yet the throats and peritoneal fluids yielded an identical coccus. We know that the Streptococcus hemolyticus sets up sporadic cases of tonsillitis and peritonitis. May not this organism, just as in the epidemics, infect the human body through milk and its products or by way of human carriers, be they handlers of infected dairy products, or those who have been in contact with cases of streptococcic angina?

Thorough clinical studies have, as a rule, not been made in these epidemics. Excluding Chapelle's investigations there is but scant mention of streptococcic peritonitis. The latter made a close study of the epidemic of septic throat which occurred in Helsingfors, Finland, in 1904. He reported eighteen cases of acute diffuse peritonitis, fourteen of which were beyond question secondary to angina of streptococcic origin. The most careful bacteriological studies were made. These proved beyond doubt that he was dealing with a streptococcus and not a pneumococcus infection. Mann[34] in his report does not mention the causes of the four deaths in the Concord, N. H., outbreak. Hamburger[35] records 9 cases of fatal peritonitis in the Baltimore epidemic. Invariably when looked for in these cases the streptococcus was found in the peritoneal fluid or venous blood at autopsy examination.

ETIOLOGIC DATA. Seven of the writer's eight cases occurred in females. Isolated cases occurring in males are recorded in the literature. Our cases were all in the young; their ages ranged from ten months to twenty-one years. All occurred during the winter and spring months. This corresponds exactly to the seasonal incidences of the streptococcic throat epidemics. Longcope and Fox[36] have

[29] Arb. a. d. Path. Inst. d. Univ. Helsingfors, 1907, Bd. ii, p. 583.
[30] Bull. Johns Hopkins Hosp., 1913, xxiv, 1.
[31] Jour. Am. Med. Assn., 1912, lviii, 1111. Capps: Jour. Am. Med. Assn., 1912, lxi, 723.
[32] Jour. Am. Med. Assn., 1912, lviii, 773.
[33] Ibid., 1912, lviii, 1852.
[34] Jour. Infect. Dis., 1913, xii, 481.
[35] Loc. cit.
[36] Bull. Ayer Clinical Lab., Pennsylvania Hosp., 1906, No. 3, i, 111.

found the Streptococcus hemolyticus occasionally in the throats of healthy individuals and have demonstrated that the virulent forms are more common in the mouths of healthy persons during the cold months. Throat cultures when done in our cases have yielded a streptococcus in pure culture.

ANGINA. In but few instances was there a history of pain in the throat or tonsillitis. By the time the patients were admitted to the hospital all evidence, except for an intensely hyperemic pharynx, were usually absent. In a few cases the gravity of the abdominal condition unfortunately caused one to pay but scant attention to the throat. Whether or not a previous exudate had been present is difficult to say. It is possible that in those cases there had been no reaction on the part of the tonsils and the streptococcus gained easy access to the blood stream. Marked enlargement of the cervical glands was absent in our series, evidence of nature's failure of defence against systemic invasion.

PERITONITIC STAGE. The history, as a rule, was that the patient had suddenly, while in the best of health, been taken sick with high fever, reaching 104° F., or over, severe generalized abdominal pain, repeated vomiting and marked prostration. These phenomena persisted until admission. When the peritonitis followed a distinct anginal attack we frequently found that a period of well-being, lasting three or four days, intervened before the sudden development of the peritonitic symptoms. The temperature remained high without marked oscillations. The pulse was invariably very rapid and out of proportion to the temperature. It was at first of a bounding character. Herpes labialis was invariably absent in our series. Chapelle records its occurrence in a few instances.

Diarrhea occurred in but few of our cases. This is in contrast to the cases reported in the literature. Diarrhea is frequently stated to have preceded the peritonitis. This fact might argue for an intestinal origin in these instances. The leukocyte count was very high, ranging between 25,000 and 64,000, on the average 40,000. The polynuclear count varied from 85 to 97 per cent., on the average 92 per cent.

The abdomen was tender and moderately rigid all over. There was no marked distention, and careful examination only infrequently revealed evidences of fluid in the flanks. The patient rapidly passed into the terminal phase of peritonitis, with the usual attendant phenomena of Hippocratic facies, livid, clammy extremities and running, thready pulse. Death occurred in from three to eight days of the onset of the peritonitic stage, and was the result of cardiac and vasomotor collapse, induced by a virulent toxemia.

Ehringer mentions three cases of subacute character which developed localized abscesses and recovered after incision and drainage. These were all of mild character, without prodromal signs, slight if any vomiting, and prompt subsidence of fever.

BLOOD CULTURE. Blood cultures were taken during life in but four of our cases. In only one was a positive culture obtained, which showed a streptococcus in pure culture. In this case a blood culture had been taken thirty-six hours after the onset of the illness. This corresponds to Libman's[37] findings of sterile cultures in his cases of acute terminating peritonitis occurring in chronic parenchymatous nephritis. Cultures would very likely yield positive results more often if they could be taken at the very inception of the peritonitic process and not on the second to fourth day after, as was unfortunately the case in our series. Chapelle in one instance recovered the streptococcus on the fifth day of the disease, which was one day after operation and two days before exitus. These instances of positive blood cultures during the course of the peritonitis are the only ones to be recorded in literature. A positive blood culture, especially very early in the course of the disease, speaks very forcibly for the hematogenous origin of the peritonitis, and cannot be ascribed to an agonal bacteremia.

PROGNOSIS AND TREATMENT. Because of the absence of any primary focus that can be surgically evacuated the prognosis must of necessity be bad. Ehringer's 3 cases of localized abscess formation recovered after operation and drainage. Five cases of Chapelle's group were operated upon and all died. In the Chicago epidemic, Capps and Miller[38] reported nine deaths from streptococcic peritonitis. They failed to mention if there were any cases that recovered. The inference is that all who developed this complication died. Early operation and drainage of the peritoneal cavity is indicated. This has resulted in the saving of 2 of our 8 cases, an unusually high percentage of recoveries. In these 2 cases the throat lesions and glands of the neck were markedly in evidence.

OPERATIVE FINDINGS. The peritoneal exudate varied from that of a thin serofibrinous to a fibrinopurulent character. Though Flexner, Manahan and Fishbein have, in the secondary peritonitides, shown that the character of the fluid is, as a rule, independent of the type of organism the peritoneal exudates of our cases were only exceptionally of the thick creamy character noticed in our primary pneumococcic cases. The peritoneal fluid yielded the Streptococcus hemolyticus in pure culture in seven cases and associated with the Bacillus coli in one case. This is highly instructive as compared with the fairly frequent finding of multiple organisms in the cases of peritonitis secondary to a lesion of an abdominal viscus. Monoinfection of the peritoneum would therefore strongly argue for a hematogenous source of infection.[39] As the Bacillus coli is seldom present in the blood infections it is highly probable that it was present as a secondary invader in the peritoneal exudate of our case.

[37] Bull. Johns Hopkins Hosp., 1906, xvii, 215.
[38] Loc. cit.
[39] Wilder: Tr. Chicago Path. Soc., 1916, x, 46.

Thorough search was made at operation for any gross visceral abdominal lesion. In no instance at operation or postmortem have we been able to find anything that could be accused of being the lesion responsible for the peritonitis; nor were there any evidences of cirrhosis, cardiac defect, chronic nephritis or other chronic disease present. These cases were therefore distinct from the terminating primary peritonitis of Flexner and closely allied if not identical with the streptococcic peritonitis found during epidemics of sore-throat.

DIFFERENTIAL DIAGNOSIS. Terminating primary peritonitis is superimposed on some chronic disease and occurs in either sex, while streptococcic peritonitis of the type herein described occurs in the previously healthy and almost exclusively in young females.

Pneumococcic peritonitis is in one-third of the cases secondary to pneumococcic lesions in the lung or pleura. The onset is stormy and the course of the disease is milder. The temperature is lower. Herpes is frequent. Subacute cases with localized peritonitis are more common, and these have a creamy, thick, peritoneal exudate. The prognosis is better. Throat cultures and peritoneal fluid show a pneumococcus on careful bacteriological examination. The streptococcic cases have a less sudden and milder onset, a prodromal period of angina, malaise and abdominal cramps. The course is very severe, nervous symptoms of toxemia are more marked and diarrhea may occur early and be persistent.

Pneumonia with pneumococcic appendicitis yields, besides the evidences of pneumonia, distinct localized rigidity and tenderness in the right lower abdomen.

Acute appendicitis, with subsequent general peritonitis, at the onset shows normal or only slightly elevated temperature. There is but slight vomiting. Diarrhea is rare. There is no marked prostration early; the pulse is then not very rapid and the rigidity for a considerable time is localized in the right iliac fossa.

Acute perforative peritonitis gives a history of a primary focus in a hollow viscus, *e. g.*, gastric or duodenal ulcer, gall-bladder disease, or intestinal lesion. The onset is marked by a very severe pain localized at the site of perforation. The temperature at the beginning is normal or subnormal. The abdomen during the first few hours is not rigid all over. The pulse-rate is not rapid until late. A secondary stage of apparent improvement occurs before the final general peritonitis supervenes. There is no diarrhea.

Acute gonococcic peritonitis gives previous symptoms and signs referable to the uterus or adnexa. Vaginal smears may yield gonococci. There is no marked abdominal rigidity. The symptoms usually abate in twenty-four hours. The great majority of cases recover. The temperature and pulse are lower. The peritoneal exudate is mostly in the pelvis and is usually of a dry fibrinous character, only occasionally purulent.

Typhoid peritonitis usually occurs late in the course of the disease. The enlarged spleen, positive Widal, typical roseola and blood culture assist in the differential diagnosis.

Localized tuberculous peritonitis is with difficulty to be distinguished from localized forms of streptococcic peritonitis.

Streptococcic sepsis is secondary to a suppurative focus in the body, is frequently complicated by acute endocarditis and blood cultures are repeatedly positive.

CASE I.—Sadie F., J. H. No. 25993, aged five years, was admitted into the service of Dr. William Linder on May 12, 1914. Two days ago she began to suffer with severe abdominal cramps, constipation, high fever and vomiting. These persisted on admission. She appeared splendidly nourished. Her cheeks were flushed. The abdomen was tender and showed a doughy rigidity all over. Temperature, 104° F., pulse, 160; respirations, 23. An enema resulted in the passage of sour smelling mucus accompanied by a few particles of feces. The urine showed a heavy trace of albumin, many hyaline and granular casts and a moderate number of red blood cells. Leukocytes, 28,000; polynuclears, 94 per cent. Operation performed the same day revealed thin, creamy pus in the peritoneal cavity, omentum congested and all abdominal viscera negative. Examination of the pus from the peritoneal cavity revealed the Streptococcus hemolyticus and Bacillus coli. The patient died five days later. No postmortem examination was permitted.

CASE II.—Annie G., J. H. No. 29330, aged ten months, was admitted December 18, 1914, into the service of Dr. William Linder. She had been sick for a few days with a high fever and marked prostration. Examination pointed to a general peritonitis. Preoperative diagnosis was pneumococcic peritonitis. Leukocytes, 60,000; polynuclears, 85 per cent. Temperature, 103.6° F.; pulse, 176; respirations, 60. Operation was performed immediately. On opening the peritoneal cavity there was an escape of a large quantity of seropurulent fluid containing flocculi. The appendix and other viscera were found normal. The child did not react and died during the night. Culture of the peritoneal fluid yielded the Streptococcus hemolyticus.

CASE III.—Gertrude K., J. H. No. 37041, aged twenty-one years, was admitted into the service of Dr. Ronsheim on April 8, 1916. Appendectomy had been performed seven years ago. Menstruation was, as a rule, irregular and at long intervals. She had given birth to one child. Last menstrual period thirty-three days before admission. On the day before her entrance to the hospital she was suddenly seized with severe cramps, high fever and vomiting. The symptoms persisted on admission. She was restless. Cervical glands were palpable. The abdomen was slightly distended, very rigid and tender throughout. Temperature, 104° F., pulse, 152; respirations, 28. Leukocytes, 20,000; polynuclears, 93 per cent.

Urine showed a faint trace of albumin and a few pus cells. Blood culture was sterile. On the mistaken diagnosis of a peritonitis, secondary to a tubal condition, a vaginal section was performed three days after admission. A large amount of thin pus escaped. This on culture yielded the Streptococcus hemolyticus. She died three days later. Thorough postmortem examination revealed no primary focus in the abdominal cavity.

Case IV.—Lillian L., J. H. No. 31352, aged ten years, was admitted April 20, 1915, into the service of Dr. William Linder. One week before admission she was taken ill with a tonsillitis, which was treated for three days. After this period she felt well for two days. Two days before admission she was taken with a chill. Abdominal cramps, vomiting, constipation and high fever set in and persisted. Her face was flushed and cyanotic. The tonsils were large and together with the pharynx markedly congested. A few cervical glands were palpable. The abdomen was distended, slightly rigid and tender all over. Temperature, 105.8° F.; pulse, 160; respirations, 40. The urine was sterile on culture and yielded a trace of albumin. Leukocytes, 44,000; polynuclears, 96 per cent. A vaginal smear was negative for gonococci. Operation done on the same day failed to reveal any evident source for the marked general peritonitis present. A large amount of thin pus containing large green flocculi was evacuated. On culture this yielded the Streptococcus hemolyticus. The patient developed intra-abdominal collections of pus which caused a protracted temperature for one month. These collections were evacuated by means of the insertion of dressing forceps into the wound. The patient made a perfect recovery and was discharged cured just two months after admission.

Case V.—Minnie E., J. H. No. 41321, aged six years, was admitted January 27, 1917, into the service of Dr. William Linder. Abdominal pain, fever and vomiting set in thirty-six hours ago. The vomiting was repeated three times. Bowels moved with enema. On admission the abdomen was seen to be distended, with generalized rigidity and tenderness. Temperature, 104° F.; pulse, 160; respirations, 40 and labored. The child was pale, drowsy and restless. Blood culture revealed the Streptococcus hemolyticus. Urine showed evidences of a marked toxic nephritis, heavy trace of albumin, many hyaline and granular casts and marked acetone. Leukocytes, 64,000; polynuclears, 92 per cent. Vaginal examination showed no gonococci. Operation was performed three and one-half hours after admission. The appendix and all other viscera except for superficial congestion were normal. A moderate amount of serofibrinous flakes were present in the pus. The peritoneal fluid on culture revealed the Streptococcus hemolyticus. The child did not do well and died on the fourth day after operation.

Case VI.—Pauline E., J. H. No. 41424, aged nineteen years, was admitted into the service of Dr. William Linder, February 3,

1917. Three weeks ago she had suffered from general abdominal pains, which were intermittent in character. There were no gastric symptoms, no chills or fever. Her bowels were constipated. Three days ago she was suddenly seized with a dizzy spell, which was followed by persistent nausea and projectile vomiting. Paroxysmal generalized abdominal cramps were present. The temperature was high, ranging between 105° and 106° F. She had a persistent diarrhea, having five or six movements daily. There was no blood in the stool. On one occasion she vomited one-half tumblerful of clotted blood. This observation was made by her physician in attendance. These symptoms persisted to admission. Examination on admission to the hospital showed marked cyanosis and a state of collapse. The abdomen was tender all over. Shifting dulness was present. The extremities were cold and clammy. The patient was menstruating at the time. Blood culture taken the same day proved sterile. The urine showed a heavy trace of albumin and many hyaline and granular casts. Leukocytes, 40,000; polynuclears, 97 per cent. Temperature ranged between 102° and 104° F.; pulse, 120 to 160; respirations, 26. Herpes was absent. Lavage of stomach brought forth large amounts of foul light-brown fluid. The patient was moribund, and at the earnest request of the family that something be done, operation was performed the next day. On opening the peritoneum a large amount of non-odorous, seropurulent fluid escaped. The appendix, tubes, ovaries, stomach, intestines and kidneys were investigated and found normal. Some pus was removed for examination and yielded a pure culture of Streptococcus hemolyticus. The next day the patient died. Complete postmortem examination was made the next day. Absolutely no trace of anything that might be interpreted as being the primary focus was found in the abdomen. Special attention was paid to the generative organs, which were found normal.

CASE VII.—Bessie A., J. H. No. 41958, aged twelve years, was admitted into the service of Dr. William Linder, March 10, 1917. She had an attack of diphtheria at three years, and since then has a persistent discharge from both ears. She suffered from repeated attacks of tonsillitis. Six days before admission she suffered from sore-throat, fever and dysphagia. She felt better the next morning. The next three days she was somewhat feverish, but was up and about and attended school. On the day before admission she was seized with generalized abdominal pain, which were most severe about the umbilicus. She was feverish, vomited and her bowels moved several times after an enema. On the day of admission these symptoms together with a moderate diarrhea were still present. Examination revealed a well-nourished girl with flushed facies. The pharynx was reddened. No exudate on the tonsils was present. Mucopus hung down the posterior pharyngeal wall. The abdomen was not distended and was markedly tender over the lower half. Rebound

tenderness (Blumberg's sign of peritoneal irritation) was present. The temperature was 104° F.; pulse, 140; respirations, 32. The urine was negative. Leukocytes numbered 23,000; polynuclears, 89 per cent. Culture of the ear discharge yielded the Bacillus pyocyaneus and a Gram-positive coccus. Operation performed the same day revealed a normal appendix. The intestines were covered over with flakes of exudate. A large quantity of seropus was present in the pelvis. Thorough investigation revealed no primary focus. The peritoneal fluid on examination showed a Streptococcus hemolyticus. Throat cultures yielded the Streptococcus hemolyticus and Staphylococcus albus. A catheterized specimen of urine before operation showed Bacillus coli. Blood culture before operation proved sterile. The temperature and pulse stayed up for the first six days after operation and then reached normal. The patient made an uneventful recovery. Rectal examination on discharge failed to reveal any pelvic exudate such as might result from a residual abscess.

CASE VIII.—Samuel E., J. H. No. 48864, was admitted April 7, 1918, into the service of Dr. J. B. Bogart. One week before admission the patient and his sister had partaken of some spoiled milk. They both vomited profusely. The girl was promptly relieved after rectal irrigations. The patient, however, developed a sore-throat and temperature of 103° F. The mother noticed that the child's abdomen became rigid. Generalized abdominal pain was complained of on the day of admission. Diarrhea was at no time present. On admission the child appeared very ill, with pinched facies, congested throat and marked generalized abdominal rigidity and tenderness. The urine was negative. The leukocytes numbered 28,800; polynuclears, 88 per cent. Temperature, 102° F.; pulse, 120; respirations, 30. Operation was performed the same day by Dr. G. I. Miller. The peritoneal cavity was filled with much seropurulent fluid. The appendix was kinked but not inflamed and was incidentally removed. Exploration revealed no primary focus. Two cigarette drains were inserted and the abdomen closed in the usual manner. The pus from the peritoneal cavity was carefully examined bacteriologically. An organism identical in its characteristics with the Streptococcus hemolyticus was recovered in pure culture. This organism showed no capsule, did not ferment inulin and did not dissolve bile. The temperature, pulse and respiration persisted high. Seven days after operation signs of pneumonia of the right upper lobe appeared. The child died four days later.

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